Alternatively, allele specific amplification technology which depends on selective PCR amplification may be used in conjunction with the instant invention. Oligonucleotides used as primers for specific amplification may carry the mutation of interest in the center of the molecule, so that amplification depends on differential hybridization (Gibbs *et al.*, *Nucl. Acids Res.* 17:2437-2448, 1989) or at the extreme 3′ end of one primer where, under appropriate conditions, mismatch can prevent, or reduce polymerase extension (Prossner, *Tib/Tech* 11:238, 1993). In addition it may be desirable to introduce a novel restriction site in the region of the mutation to create cleavage-based detection (Gasparini *et al.*, *Mol. Cell Probes* 6:1, 1992). It is anticipated that in certain embodiments amplification may also be performed using Taq ligase for amplification (Barany, *Proc. Natl. Acad. Sci. USA* 88:89, 1991). In such cases, ligation will occur only if there is a perfect match at the 3′ end of the 5′ sequence making it possible to detect the presence of a known mutation at a specific site by looking for the presence of absence of amplification.

The methods described herein may be performed, for example, by utilizing pre-packaged diagnostic kits comprising at least one probe nucleic acid or antibody reagent described herein, which may be conveniently used, for example, in a clinical setting to diagnose patient exhibiting symptoms or a family history of a disease or disorder involving abnormal GLUTX activity.

XI. Pharmacogenetics

Agents or modulators which have a stimulatory or inhibitory effect on GLUTX activity (including those that alter activity by altering GLUTX gene expression),

identified by a screening assay described herein, can be administered to individuals to treat, prophylactically or therapeutically, disorders associated with aberrant GLUTX activity. In conjunction with such treatment, the pharmacogenetics (*i.e.*, the study of the relationship between an individual's genotype and that individual's response to a foreign compound or drug) of the individual may be considered. Thus, the pharmacogenetics of the individual permits the selection of effective agents (*e.g.*, drugs) for prophylactic or therapeutic treatments based on a consideration of the individual's genotype. Such pharmacogenetics can further be used to determine appropriate dosages and therapeutic regimens. Accordingly, the activity of GLUTX polypeptides, expression of GLUTX nucleic acids, or sequence of GLUTX genes in an individual can be determined and used to thereby select an appropriate agent for therapeutic or prophylactic treatment of the individual.

Pharmacogenetics deals with clinically significant hereditary variations in the response to drugs due to altered drug disposition and abnormal action in affected persons (See, *e.g.*, Eichelbaum, *Clin. Exp. Pharmacol. Physiol.* 23:983-985, 1996 and Linder, *Clin. Chem.* 43:254-266, 1997). In general, two types of pharmacogenetic conditions can be differentiated. Genetic conditions transmitted as single factors altering the way drugs act on the body (altered drug action) or genetic conditions transmitted as single factors altering the way the body acts on drugs (altered drug metabolism). These pharmacogenetic conditions can occur either as rare defects or as polymorphisms. For example, glucose-6-phosphate dehydrogenase deficiency (G6PD) is a common inherited enzymopathy in which the main clinical complication is

hemolysis after ingestion of oxidant drugs (anti-malarials, sulfonamides, analgesics, nitrofurans) and consumption of fava beans.

As an illustrative embodiment, the activity of drug metabolizing enzymes is a major determinant of both the intensity and duration of drug action. The discovery of genetic polymorphisms of drug metabolizing enzymes (*e.g.*, N-acetyltransferase (NAT2) and cytochrome P450 enzymes CYP2D6 and CYP2C19) has provided an explanation as to why some patients do not obtain the expected drug effects or show exaggerated drug response and serious toxicity after taking the standard and safe dose of a drug. These polymorphisms are expressed in two phenotypes in the population, the excessive metabolizer (EM) and poor metabolizer (PM). The prevalence of PM is different among different populations. For example, the gene coding for CYP2D6 is highly polymorphic and several mutations have been identified in PM, which all lead to the absence of functional CYP2D6. Poor metabolizers of CYP2D6 and CYP2C19 quite frequently experience exaggerated drug response and side effects when they receive standard doses. If a metabolite is the active therapeutic moiety, PM show no therapeutic response, as demonstrated for the analgesic effect of codeine mediated by its CYP2D6-formed metabolite morphine. The other extreme is the so called ultra-rapid metabolizers who do not respond to standard doses. Recently, the molecular basis of ultra-rapid metabolism has been identified to be due to CYP2D6 gene amplification.

Thus, the activity of GLUTX polypeptide, expression of GLUTX nucleic acid, or the precise sequence of a GLUTX gene in an individual can be determined and used to select an appropriate agent for therapeutic or prophylactic treatment of the individual. In addition, pharmacogenetic